

ABSTRACT FOR EXPERT-PE

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Background

Chest pain and dyspnoea are among the most common presenting complaints in the Emergency Department (ED) and may reflect a broad spectrum of underlying conditions. Identifying the right aetiology can be particularly challenging in patients with multiple comorbidities, since in these situations different pathological processes may coexist.

Case presentation

We report the case of a 76-year-old man with a history of arterial hypertension, dyslipidemia, type 2 diabetes mellitus, obstructive sleep apnea syndrome (OSAS), and obesity who presented to the ED with dyspnoea. Initial electrocardiography (ECG) and transthoracic echocardiography were inconclusive, while laboratory investigations revealed elevated cardiac troponin levels (0.250 µg/L). Given the high pre-test probability of ischemic heart disease, coronary angiography with coronary flow reserve (CFR) assessment was performed the following day, leading to a diagnosis of coronary microvascular dysfunction (CMD). The patient was discharged after four days.

Ten days later, he was readmitted to the ED because of worsening dyspnoea. ECG showed a minor nonspecific right-sided conduction abnormality. Cardiac troponin levels were mildly elevated (0.04 µg/L; upper reference limit, 0.014 µg/L), although the findings were not considered diagnostic of acute coronary syndrome. Chest computed tomography (CT) revealed extensive pulmonary embolism (PE). Echocardiography showed mildly increased right ventricular dimensions with preserved systolic function (TAPSE 24 mm; S' 12 cm/s) and reduced pulmonary flow acceleration time with a midsystolic notch. The simplified Pulmonary Embolism Severity Index (sPESI) was 1. Venous ultrasonography demonstrated completely occlusive thrombosis of the left femoropopliteal venous axis.

Despite the intermediate-low-risk classification, intravenous unfractionated heparin (UFH) was initiated, and the patient underwent catheter-based pulmonary thrombectomy using the Inari FlowTrier system, without procedural complications. An inferior vena cava (IVC) filter was also placed in the setting of extensive proximal deep vein thrombosis. UFH was subsequently replaced with apixaban 5 mg BID. In addition, tirzepatide was initiated during hospitalization.

At 3- and 6-month follow-up, the patient reported good functional capacity and a weight reduction of approximately 15 kg. Follow-up chest CT showed no residual pulmonary arterial emboli. The common femoral veins, iliac veins, and IVC were patent, with no evidence of thrombosis in the filter-bearing venous system. IVC filter retrieval was therefore scheduled. Follow-up echocardiography confirmed preserved right ventricular systolic function (TAPSE 25 mm, S' 11 cm/s, fractional area change 40%), with a right-to-left ventricular diameter ratio of 0.75 and an eccentricity index of 1.0. Mild tricuspid regurgitation was present, while the tricuspid regurgitation pressure gradient could not be measured. In addition, as part of the investigation for potential underlying causes of venous thromboembolism, abdominal CT revealed an 8-mm

hypervascular lesion in the pancreatic tail, raising suspicion of a pancreatic neuroendocrine tumor (NET). Further characterization with PET was recommended.

Discussion and conclusions

Our clinical case raises several interesting questions regarding the aetiology of pulmonary embolism, the selection of patients for catheter-based treatment, and the optimal duration of anticoagulation.

First, the underlying cause of venous thromboembolism remains uncertain. Several potentially contributing factors can be identified, including obesity, recent hospitalization for myocardial infarction with non-obstructive coronary arteries (MINOCA), and potentially reduced mobility following discharge. However, the incidental detection of a small hypervascular pancreatic lesion raises the possibility of an underlying neoplastic process, which requires further investigation. Clarifying whether the pulmonary embolism was associated with transient provoking factors, persistent risk factors, or an underlying malignancy is essential to guide the duration of anticoagulation in the extension period. Finally, this case highlights the importance of an individualized approach to pulmonary embolism management, emphasizing the careful assessment of eligibility for catheter-based interventions, irrespective of the European risk classification.